to cTnI at H12 (r = 0.42, p < 0.001). Major CE rate was not increased in high CRP Pts (16% vs 19%), and angiographic findings were not significantly different.

	cTnI+	cTnI–	p
CRP H0 (mg/L)	8 ± 12	6 ± 13	NS
CRP H12 (mg/L)	9 ± 13	7 ± 13	NS
CRP H24 (mg/L)	14 ± 15	7 ± 11	0.009
CRP H72 (mg/L)	17 ± 14	8 ± 11	0.002
Δ CRP (mg/L)	6 ± 10	1 ± 6	0.002

cTnI is a better clinical and angiographic prognostic marker in UA than CRP. The delayed increased of CRP suggests that systemic inflammation is a marker rather than cause of Major CE. The increase in CRP levels may partly be a consequence of myocardial cell damage.

970-40 Potentiation of Human Vascular Smooth Muscle Cell Growth by Pericardial Fluids From Patients With Unstable Angina

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The strong association between regional tissue ischemia and collateral growth continues to be a paradigm. We hypothesized that angiogenic growth factors are produced by ischemic cardiac tissue, diffusible, more concentrated in pericardial fluids, and accelerate the growth of vascular smooth muscle cells (VSMC). Pericardial fluids of 11 patients with unstable angina (group 1) and of 7 patients with nonischemic heart disease (group 2) were collected at the time of open heart surgery. VSMC cultures were established by an explant method using human internal mammary arteries. Cells were plated at the third passage at 10^4/ml and allowed to attach for 24 hr. The 7 day growth assay was preceded by 72 h of growth arrest with 0.4% fetal calf serum (FCS). Growth was then restarted by addition of medium with 10% FCS and 0.05 ml pericardial fluids of 18 patients. Cell counts on triplicate wells were done on days 0, 3, and 7 on a Coulter counter. The concentration of basic fibroblast growth factor (bFGF) in pericardial fluids was also measured with use of enzyme-linked immunosorbent assay. The effect of pericardial fluids on the growth of VSMC was presented as a ratio (R) of cell numbers of day 7 to day 0. In control culture, R was 2.14. R in group 1 was 4.24 ± 1.11 (SD), being significantly (P = 0.04) higher than 2.51 ± 0.21 in group 2. The concentration of bFGF in pericardial fluids in group 1 was 1460 ± 1251 pg/ml, being significantly (P = 0.002) higher than 318 ± 196 pg/ml in group 2. These findings provide a new evidence of a close link between myocardial ischemia and collateral development in ischemic patients.

970-41 Angiotensin-Converting Enzyme Genotype Does Not Influence Myocardial Hypertrophy After Acute Myocardial Infarction

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The homozygous angiotensin-converting enzyme (ACE) deletion-type (*D*) allele has been associated with higher plasma ACE activity, left ventricular (LV) hypertrophy, and, in post-myocardial infarction (MI) patients (pts), an augmented neurohormonal activation and left ventricular dilatation. We hypothesized that post-MI pts with *ACE DD* genotype have a greater attenuation of LV mass and volumes when treated with ACE inhibitor therapy. Forty-six pts were evaluated 1 week after an acute Q-wave MI: pts with an LV ejection fraction (EF) <40% received ramipril (n = 20) while pts with LVEF > 40% were randomized to ramipril (n = 14) or no ACE inhibitor (n = 12). Magnetic resonance imaging was performed 1 week and 3 months post-MI to determine LV mass and LV end-diastolic volume (EDV) from summated serial short axis images. There were no differences in baseline LV mass, LV EDV or LVEF in these patient groups. The allele frequencies were 0.39 for *I* and 0.61 for *D;* genotype frequencies were in agreement with Hardy-Weinberg equilibrium (p = 0.7). After 3 months of therapy, ramipril significantly decreased LV mass in all pts (p = 0.04), independent of *ACE* genotype. LV EDV, LVEF, and mass/LV EDV ratio were unaffected by either ramipril therapy or *ACE* genotype. Thus, *ACE* genotype does not influence changes in LV mass in pts treated with the ACE inhibitor ramipril during the first 3 months post-MI.

970-42 Concordance Between Troponin T and Troponin I Values in 491 Patients with Unstable Coronary Syndrome. A TRIM-substudy

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Introduction: It is known that around one-third of patients with unstable angina pectoris (UAP) with increased serum level of cardiac troponin T (cTnT) have a poor prognosis similar to those with acute myocardial infarction (AMI). Several studies support the assumption that these patients have significant myocardial damage (microinfarction). The aim of this study was to elucidate whether cardiac troponin I (cTnI) and cTnT identify the same patients with myocardial damage.

Materials and Methods: 491 consecutive patients with unstable coronary syndrome participating in the TRIM-multicenter study were investigated. Blood samples were drawn at inclusion, 6, 12 and 24 h. We analyzed for cTnT and cTnI in plasma. The cut-off values used are: cTnT 0.20 μg/l and cTnI 2.0 μg/l.

Results:

Concordance between cTnT an d cTnI values in 491 patients:

	Number of patients	Median μg/l	Range μg/l
cTnT ≥ 0.20 μg/l	217 (44%)	1.11	0.20–30.00
cTnI ≥ 2.00 μg/l	212 (43%)	10.55	2.13–232.00

Both markers were elevated in 200 patients (40%) and 92% of patients with elevated cTnT also has elevation of cTnI. In 17 patients cTnT was elevated and cTnI within normal range. In 12 patients only cTnI was elevated.

Conclusion: The majority of patients with elevated values of cTnT also have elevated values of cTnI.

970-43 Cardiac Troponin T in Patients with Renal Failure

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In patients with normal renal function cardiac troponin T (cTnT) is a sensitive and specific marker of myocardial cell injury. However, in patients with renal failure the serum concentration of cTnT may be increased without apparent cardiac ischemia. The reason for the increased serum levels of cTnT in renal failure patients is not clear. We measured cTnT in 97 patients with end stage renal disease (ESRD) with the standard and a new cTnT sandwich ELISA which uses 2 cardiac-specific anti-cTnT monoclonal antibodies. The patients were stratified into 3 groups: proven coronary artery disease (CAD) by angiography or prior myocardial infarction (*A*), 2 or more recognized cardiac risk factors (*B*), 1 or no cardiac risk factors (*C*). In each of the 3 groups the cTnT values measured with the new ELISA were significantly lower than the concentrations measured with the standard assay. In both assays the cTnT concentration correlated with (the risk of) CAD, since the mean cTnT concentration (ng/ml) in group C was significantly lower than in group A or B. In contrast, there was no significant difference in the serum activity of CK between the groups (all values x ± SEM):

	A (n = 22)	B (n = 48)	C (n = 27)
cTnT, stand. ELISA	0.45 ± 0.15	0.39 ± 0.12	0.12 ± 0.03*
cTnT, spec. ELISA	0.26 ± 0.08	0.23 ± 0.06	0.07 ± 0.02*
CK (U/l)	20.8 ± 2.7	21.6 ± 2.4	16.2 ± 2.6

We also examined cTnT expression in skeletal muscle biopsies of ESRD and control patients. cTnT was not detected in skeletal muscle at the protein (immunoblot, immunofluorescence) or RNA level (reverse transcribed PCR). *Conclusions:* The increased serum concentration of cTnT in patients with ESRD is not explained by the expression of cTnT in skeletal muscle. The chronically increased cTnT concentration in the ESRD population correlates with cardiac risk. The second generation ELISA with increased specificity for cTnT is the assay of choice in patients with renal failure.

970-44 Plasma levels of soluble adhesion molecules VCAM-1 and L-selectin during acute myocardial infarction

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Background: Adhesion of activated neutrophils (PMN) to endothelial cells during acute myocardial infarction may reduce coronary capillary flow and thus contribute to myocardial injury. PMN adhere to the endothelium via